

CONTINUING EDUCATION PROGRAM: FOCUS...

Infectious and metabolic brain imaging

E. Auffray-Calvier*, F. Toulgoat, B. Daumas-Duport,
A. Lintia Gaultier, H. Desal

*Diagnostic and Interventional Neuroradiology Department, Hôpital René et
Guillaume-Laënnec, CHU de Nantes, 44800 Saint-Herblain, France*

KEYWORDS

Brain;
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Abstract Central nervous system infectious and metabolic disease is a vast domain. We have chosen to focus particularly on five pathological conditions: brain abscess, herpes encephalitis, Creutzfeldt-Jacob disease, posterior reversible encephalopathy and central pontine myelinolysis. We will pay particular attention to MRI signs and the specific sequences to use in each condition, in addition to the conventional sequences, in order to avoid diagnostic traps. Once the MRI exploration is complete, the diagnosis still cannot be established without knowing the clinical and metabolic context.

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Since the number of infectious and metabolic brain diseases is very great and the diseases very varied, we have chosen here to present those that seem most likely to be wrongly diagnosed. We will therefore consider five pathological conditions: brain abscess, herpes encephalitis, Creutzfeldt-Jacob disease, posterior reversible encephalopathy and central pontine myelinolysis. The objective is to have good knowledge of the clinical situation and the MRI signs, including the different sequences that are useful for establishing the best diagnosis in the following situations:

- an abscess versus a haemorrhagic lesion, metastasis or brain tumour;
- herpes encephalitis versus a brain tumour, status epilepticus, an ischaemic stroke, limbic encephalitis and syphilitic encephalitis;
- Creutzfeldt Jakob disease versus metabolic encephalopathy or cerebral anoxia, deep vein thrombosis and status epilepticus;

* Corresponding author.

E-mail address: elisabeth.calvier@chu-nantes.fr (E. Auffray-Calvier).

- posterior reversible encephalopathy syndrome (PRES) versus vasculitis, venous thrombosis and progressive multifocal encephalopathy;
- central pontine myelinolysis versus brain stem ischaemia, PRES and leukoaraiosis.

Brain abscess

A brain abscess will be suspected when there is a fluid-filled lesion surrounded by oedema. The clinical context — postoperative, traumatic, sinusitis, endocarditis or immunosuppression — can sometimes provide us with a pointer. Samples will be taken to find the causative organism.

MRI signs

A brain abscess of bacterial origin is a fluid-filled lesion surrounded by oedema; the centre is hypointense with T1-weighting and hyperintense with T2/FLAIR. Its wall is hyperintense with T1-weighting and hypointense with T2/T2*. Parietal enhancement after gadolinium injection is thin and regular (Fig. 1a–c). Although these signs are evocative, the diagnosis is based mainly on the data from the diffusion sequence. A pyogenic abscess typically shows diffusion restriction and a fall in the apparent diffusion coefficient (ADC) in contrast to observations in the case of a necrotic tumour [1] (Fig. 1d–e). With infectious collections there is no increase in cerebral blood volume (CBV) in the wall (Fig. 2a–f). Finally, in proton spectroscopy, with a short echo time (TE), an amino-acid multiplet is seen centred at

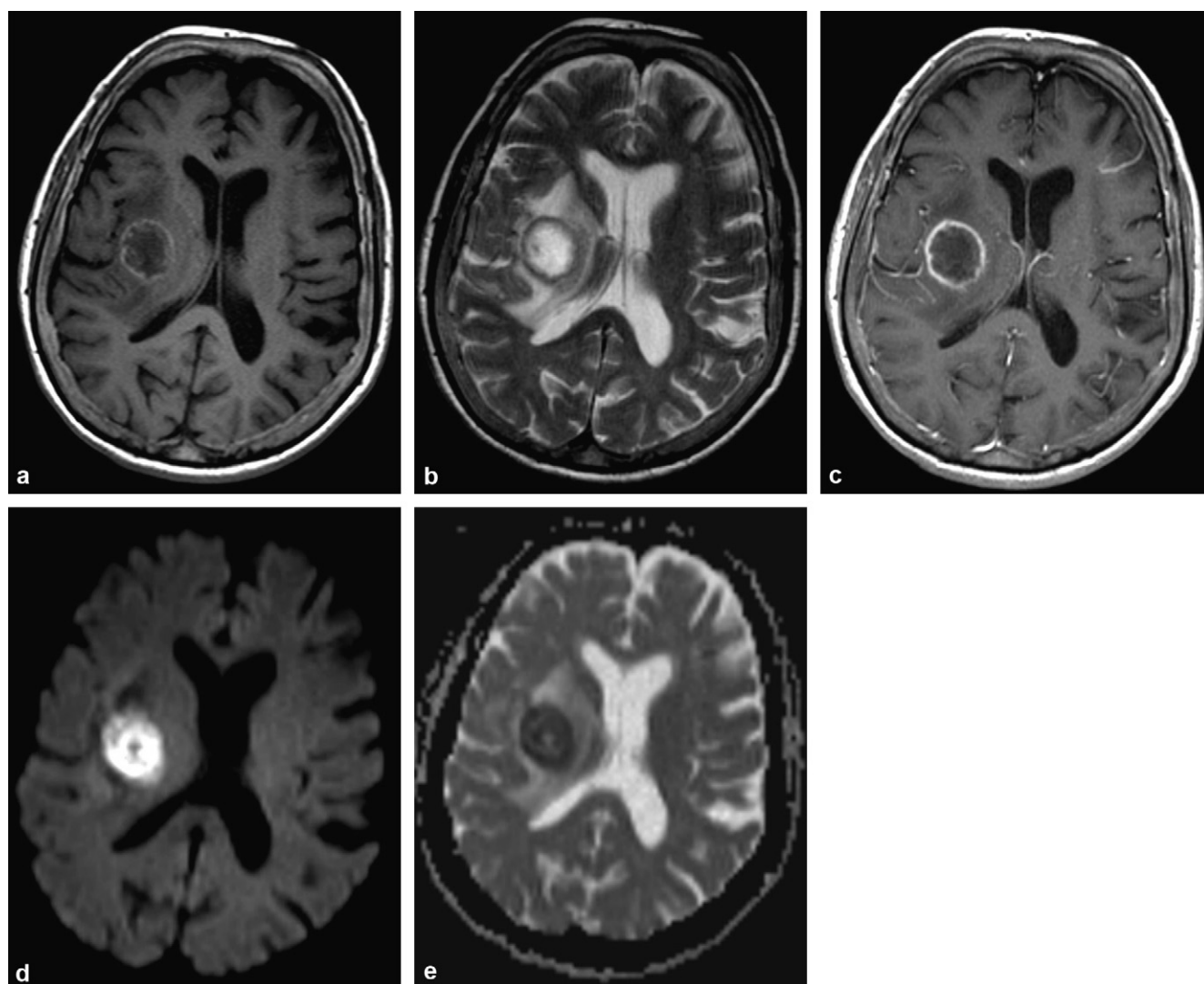


Figure 1. Right cerebral abscess, typical MRI appearance. a: T1-weighted axial slice: hyperintense wall and hypointense centre; b: T2-weighted axial slice: hypointense wall, hyperintense centre and peripheral oedema; c: T1-weighted axial slice after gadolinium injection: thin wall enhancement; d: diffusion-weighted axial slice: hyperintense; e: apparent diffusion coefficient (ADC) mapping: ADC reduction in the collection.

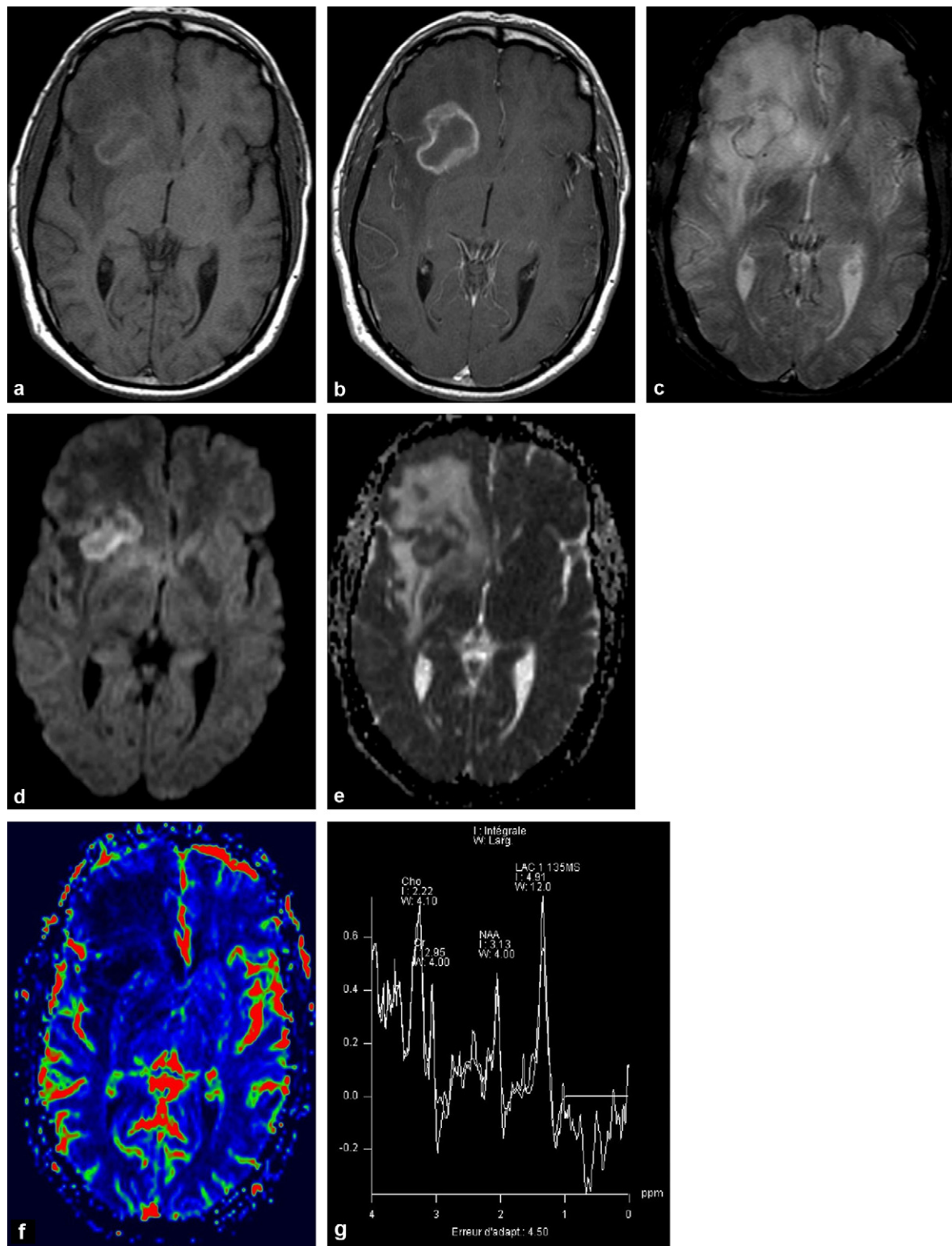


Figure 2. Atypical abscess: right frontal-temporal expansive lesion. a: T1-weighted axial slice: heterogeneous lesion with spontaneous high intensity signal from the wall; b: T1-weighted axial slice after gadolinium injection: irregular dense enhancement of the wall; c: T2*-weighted axial slice: hypointense wall and considerable perilesional oedema; d: diffusion-weighted axial slice: hyperintense signal from the lesion predominantly from the wall; e: axial apparent diffusion coefficient (ADC) mapping: wall ADC reduced; f: perfusion sequence: no increase in CBV; g: Proton spectroscopy sequence with long echo time (TE) (135 ms): amino-acid multiplet negative at 0.9 ppm suggesting a cerebral abscess.

0.9 ppm inverting at long TE (TE: 135 ms) [2] (Fig. 2g). These spectroscopic data are complementary and can be of great importance in atypical cases with thick and irregular contrast enhancement of the wall. A haemorrhagic lesion can however be misleading because the blood breakdown products can interfere with the diffusion sequence. They are sometimes responsible for hyperintensity in diffusion and a fall in ADC. These haemorrhagic lesions will be clearly visible as hypointense areas on the T2*-weighted sequence and if it is a haemorrhagic tumour, the increased CBV will point towards a high-grade tumour (Fig. 3). In addition, although rare, it is possible to observe diffusion restriction in necrotic metastases [3] (Fig. 4). However, not all abscesses show diffusion restriction, as in the case of the toxoplasma abscess, which can show variable behaviour and often displays an absence of restriction. This aetiology must be considered in immunosuppressed patients [4] (Fig. 5).

Exploration protocol when a brain abscess is suspected

Exploration protocol when a brain abscess is suspected:

- T1-weighting: spontaneous hyperintensity of the wall;
- T2/FLAIR-weighting: central hyperintensity;
- T2*-weighting: hypointensity of the wall. This sequence will look for a haemorrhagic lesion which would disrupt diffusion;
- Diffusion+++: a fall in the ADC in the fluid-filled lesion is highly suggestive of a pyogenic abscess;
- perfusion: no increase in CBV occurs in the case of an abscess;
- T1-weighting + gadolinium: fine enhancement of the wall;
- T1-weighted 3D neuro-navigation: for a diagnostic biopsy;
- proton spectroscopy: if a diagnostic doubt remains, the presence of amino acids will be highly suggestive of an abscess.

Herpes encephalitis

Bilateral but asymmetrical involvement of the limbic system in a patient with a picture of encephalitis involving headaches, consciousness and behavioural disorders, epileptic attacks (olfactory, auditory, or psychomotor), aphasia and hyperthermia, should, until proven otherwise, suggest a diagnosis of herpes encephalitis (HE). A PCR herpes test will confirm the diagnosis.

MRI signs

In T2/FLAIR-weighting, herpes encephalitis shows as bilateral, asymmetrical, discretely expansive, cortical-subcortical hyperintensity, selectively affecting the limbic system (hippocampus, medial temporal cortex, insula, ventral frontal cortex, cingulate cortex) [5] (Fig. 6a–b). A T2*-weighted sequence is very sensitive for detecting haemorrhagic necrosis which appears hypointense (Fig. 6c). With T1-weighting, the lesion is hypointense. After gadolinium injection, enhancement is possible and most often cortical and gyriform. At the onset of symptoms, a diffusion sequence is the most sensitive and can reveal a fall in the ADC [6,7] (Fig. 7). In the early stage, involvement can be

unilateral and wrongly suggest a tumour, status epilepticus or an ischaemic stroke. If ischaemia is suspected and the clinical history is misleading, MR angiography will correct the diagnosis (Fig. 8) by revealing a stenosis or occlusion. In HE, there is no lenticulo-caudate involvement and a thalamic lesion is rarely present [8]. The existence of a lenticular or caudate extension points towards limbic encephalitis [9] (Fig. 9). Finally, where there is subacute development of deterioration to cognitive functions, a diagnosis of syphilitic encephalitis should be considered, in which a classic sign is a preponderance of lesions in the temporal lobes [10] (Fig. 10).

Exploration protocol when herpes encephalitis is suspected

Exploration protocol when HE is suspected:

- T2/FLAIR-weighting: bilateral but asymmetric involvement of the limbic system with the basal ganglia unaffected;
- T1-weighting before and after injection: gyriform enhancement is possible in HE;
- T2*-weighting: haemorrhagic changes are common;
- diffusion: at the onset of symptoms, this is the most sensitive sequence.

If involvement is unilateral:

- MR angiography: will look for evidence indicating a stenosis or arterial occlusion, the presence of which will point towards a diagnosis of ischaemia;
- a perfusion sequence: will point towards a brain tumour if there is an increase in CBV.

Creutzfeldt-Jakob disease

This diagnosis will be suggested when there are bilateral striatal and/or cortical lesions in a context of rapid deterioration of higher functions often associated with myoclonus. Pulvinar involvement related to the new variant is rarer. The diagnosis of 'probable' sporadic Creutzfeldt-Jakob disease (CJD) is based on the presence of the 14.3.3 protein in the CSF. Diagnosis with certainty will be established from the neuropathological data with evidence found of the pathological prion protein in the brain [11].

MRI signs

Lesions related to CJD are found in the grey matter: the striatum, cortex and pulvinar [12,13]. The lesions are not visible with T1-weighting and there is no enhancement after contrast injection. T2/FLAIR-weighted sequences show non-expansive hyperintensity (Fig. 11). When the symptoms have only recently started, diffusion is the most sensitive sequence, and will show a fall in the ADC [14,15] (Fig. 12). However, when faced with involvement of both nuclei and the cortex, the possibility of a metabolic or anoxic condition should be discussed. In these situations the clinical context very often provides clues [16] (Fig. 13). The existence of haemorrhagic changes will lead to an MR venography examination to look for deep vein thrombosis [17] (Fig. 14). Finally, cortical involvement associated with pulvinar lesions can be

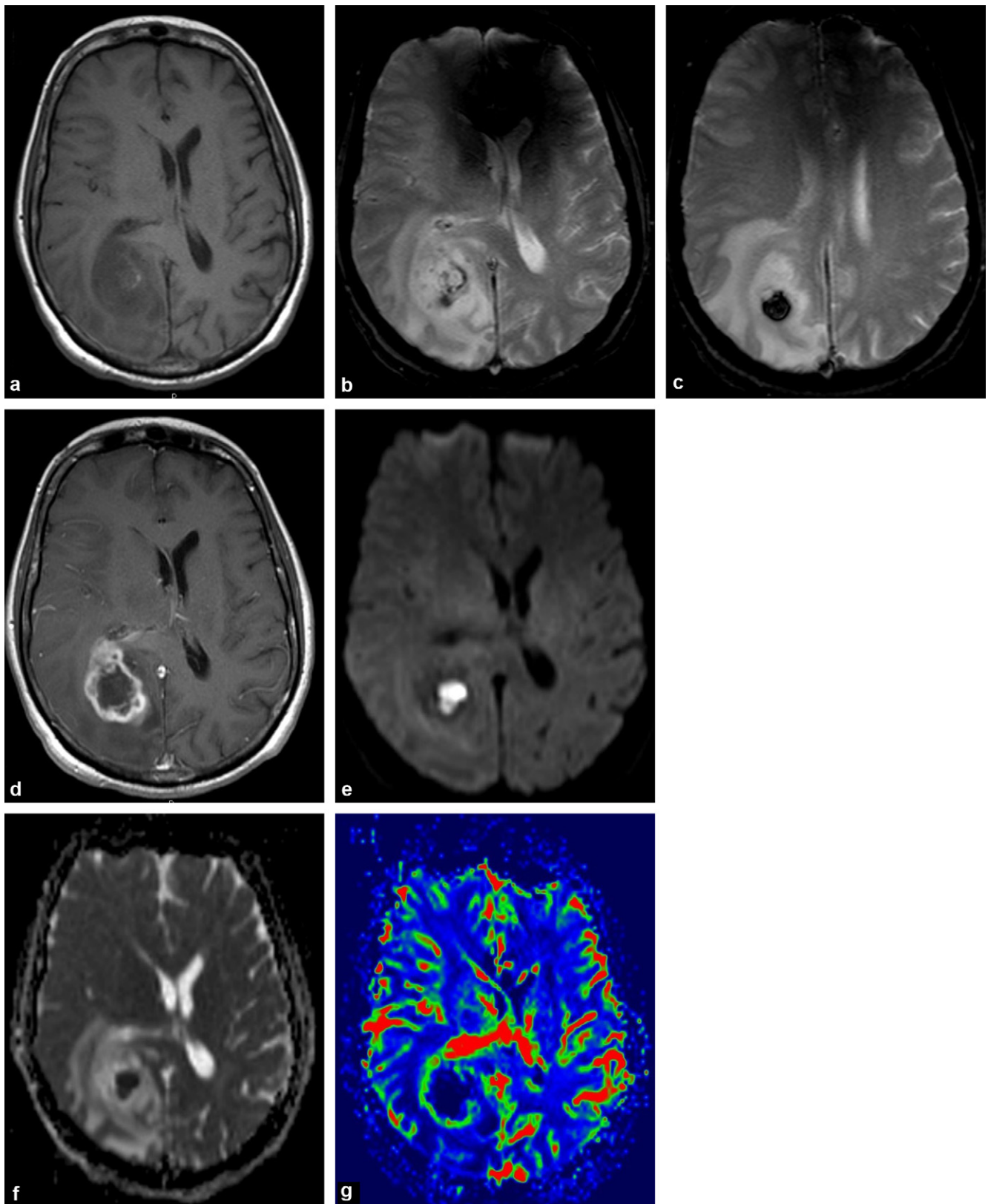


Figure 3. Right parietal-occipital glioblastoma: the haemorrhagic component is responsible for a fall in apparent diffusion coefficient (ADC). a: T1-weighted axial slice: heterogeneous lesion with spontaneous hyperintensity; b–c: T2*-weighted axial slices: marked hypointense haemorrhagic changes; d: T1-weighted axial slice after gadolinium injection: irregular dense enhancement of the necrotic lesion; e: axial diffusion-weighted sequence: central hyperintensity; f: axial ADC mapping: reduced ADC due to haemorrhage; g: axial perfusion sequence: CBV increased in the wall suggesting a high-grade tumour.

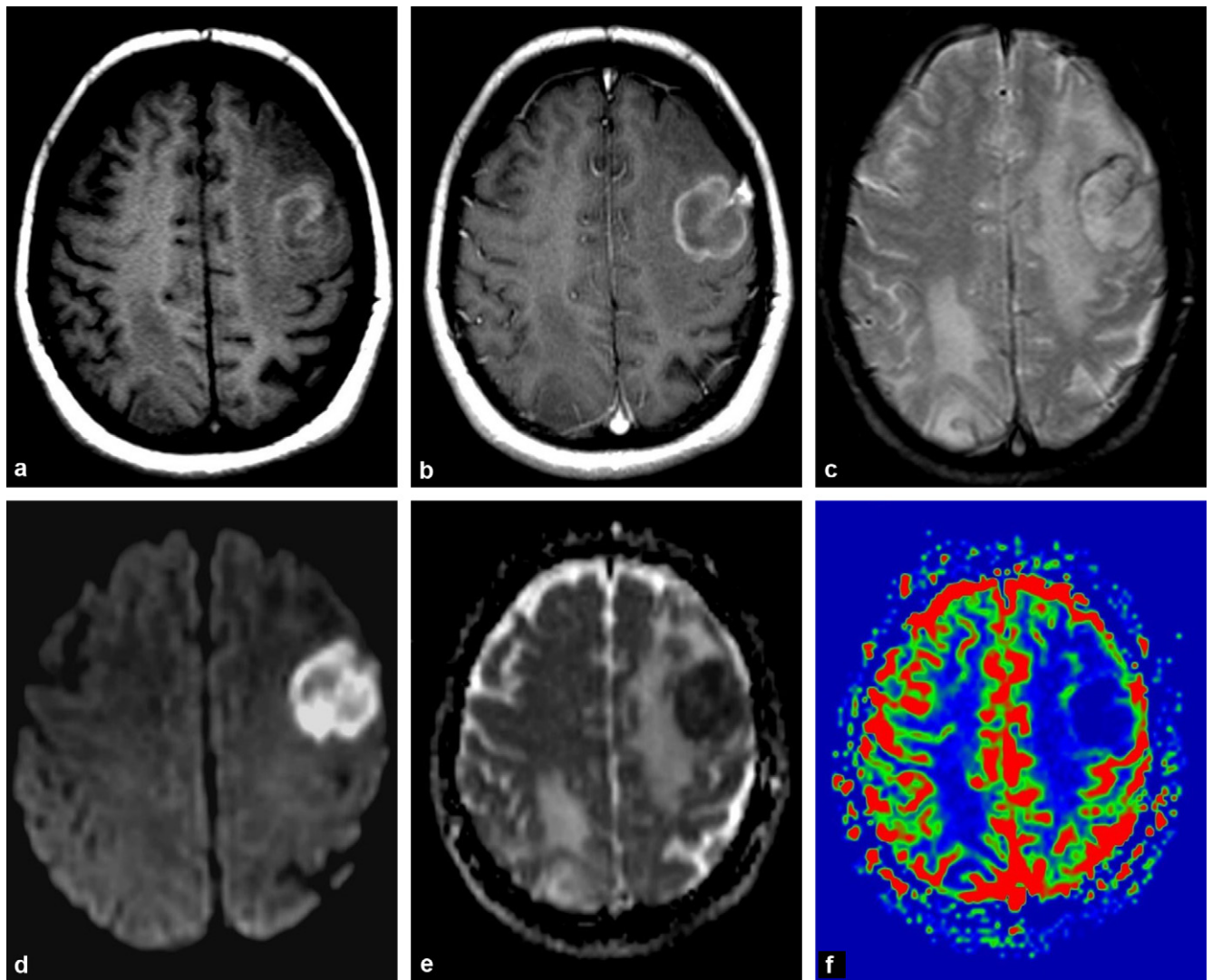


Figure 4. Left frontal necrotic metastasis with diffusion restriction. Second right parietal lesion. a: T1-weighted axial slice: heterogeneous lesion; b: T1-weighted axial slice after gadolinium injection: thin enhancement of the wall; c: T2*-weighted axial slice: hypointense wall and hyperintense centre. There is a 2nd right parietal lesion; d: diffusion-weighted axial slice: the entire lesion is hyperintense; e: axial apparent diffusion coefficient (ADC) mapping: ADC reduced; f: axial perfusion sequence: CBV increased on the inner side of the wall.

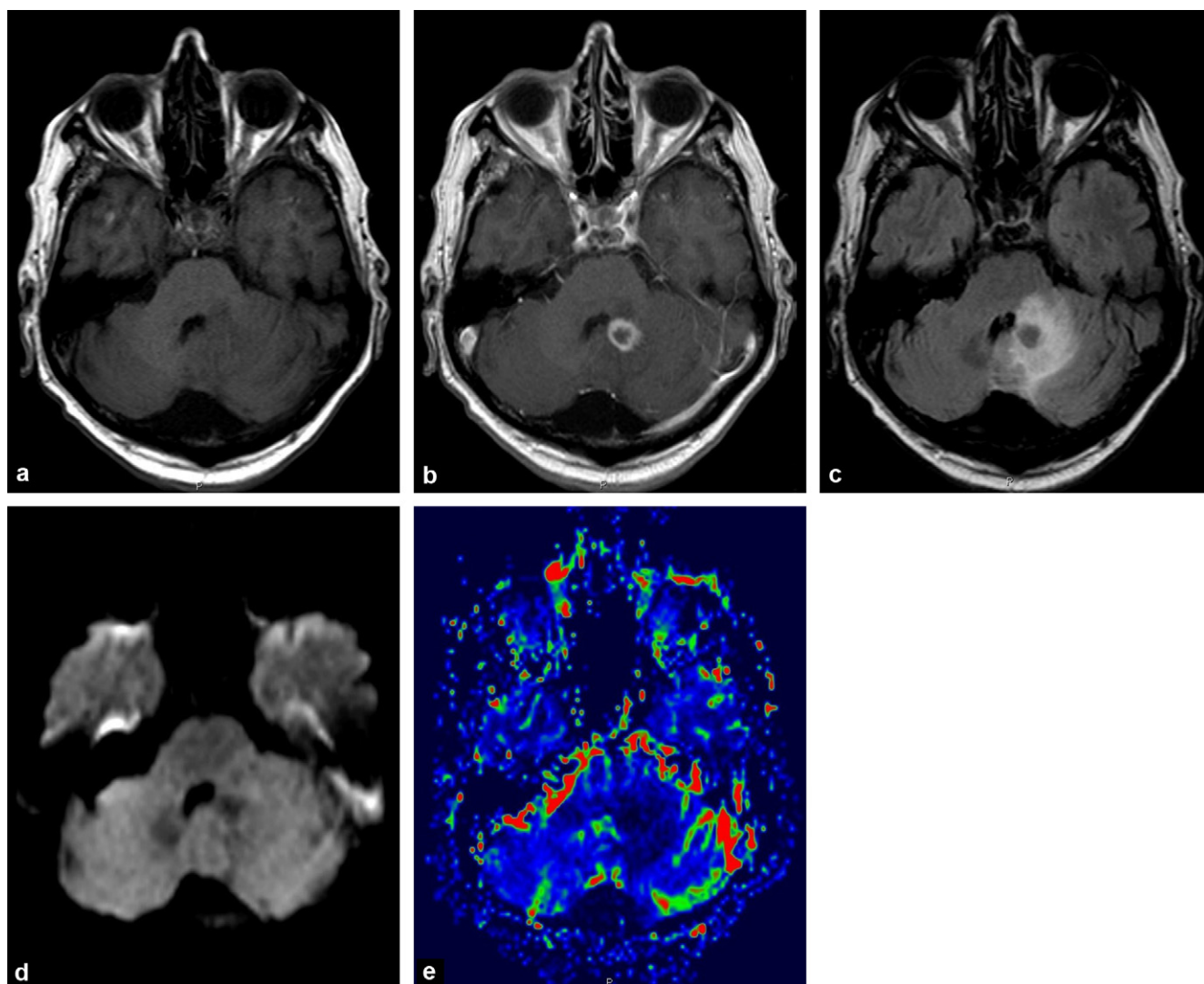


Figure 5. Left cerebellar toxoplasma abscess in an immunosuppressed patient. a: T1-weighted axial slice: hypointense lesion; b: T1-weighted axial slice after gadolinium injection: regular enhancement of the wall; c: FLAIR-weighted axial slice: hypointense lesion and hyperintense peripheral oedema; d: diffusion-weighted axial slice: hypointense lesion; e: axial perfusion sequence: no increase in CBV.

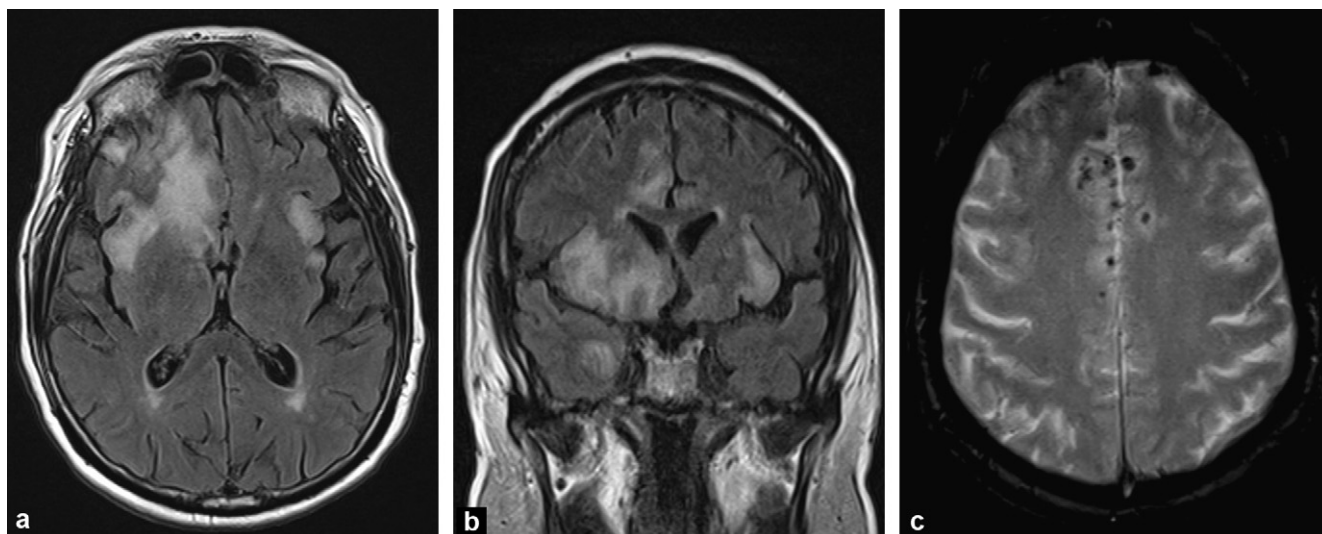


Figure 6. Herpes encephalitis with haemorrhagic cortical necrosis in a 64-year-old man with febrile confusion. a–b: FLAIR-weighted axial and coronal slices: bilateral frontal-temporal-insular hyperintensity predominantly on the right; c: T2*-weighted axial slice: frontal cortical hypointensity caused by haemorrhagic necrosis.

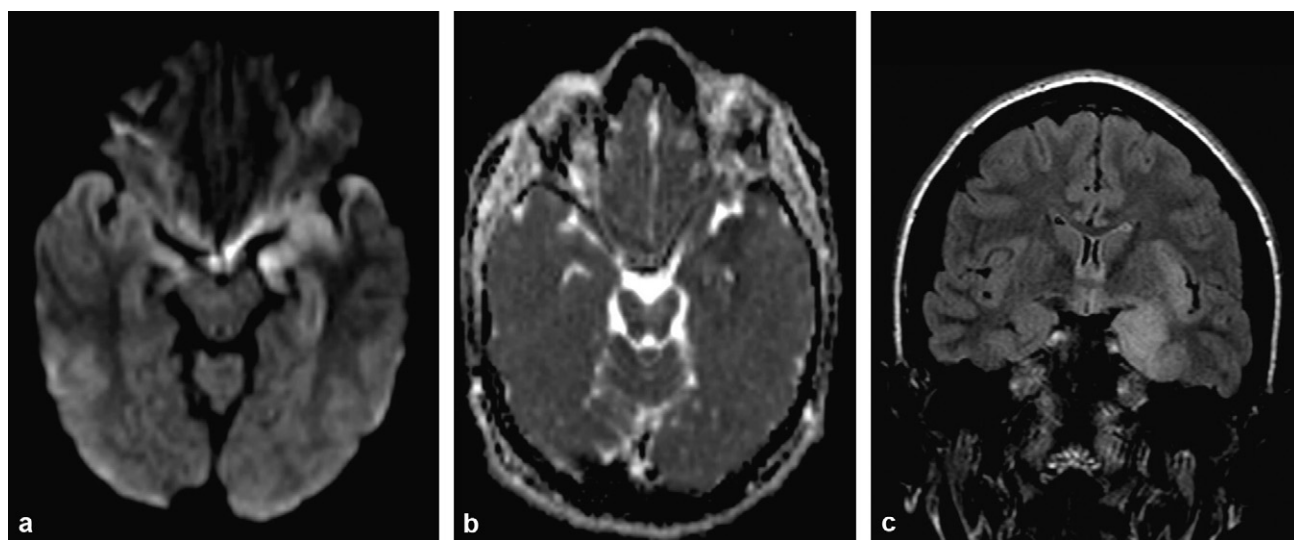


Figure 7. Herpes encephalitis, early MRI in a 31-year-old woman hospitalised for febrile delirium and aphasia that started 4 days previously. a: Diffusion-weighted axial slice: left temporal hyperintense lesion; b: axial apparent diffusion coefficient (ADC) mapping: ADC reduced; c: FLAIR-weighted coronal slice: left temporal-insular hyperintensity.

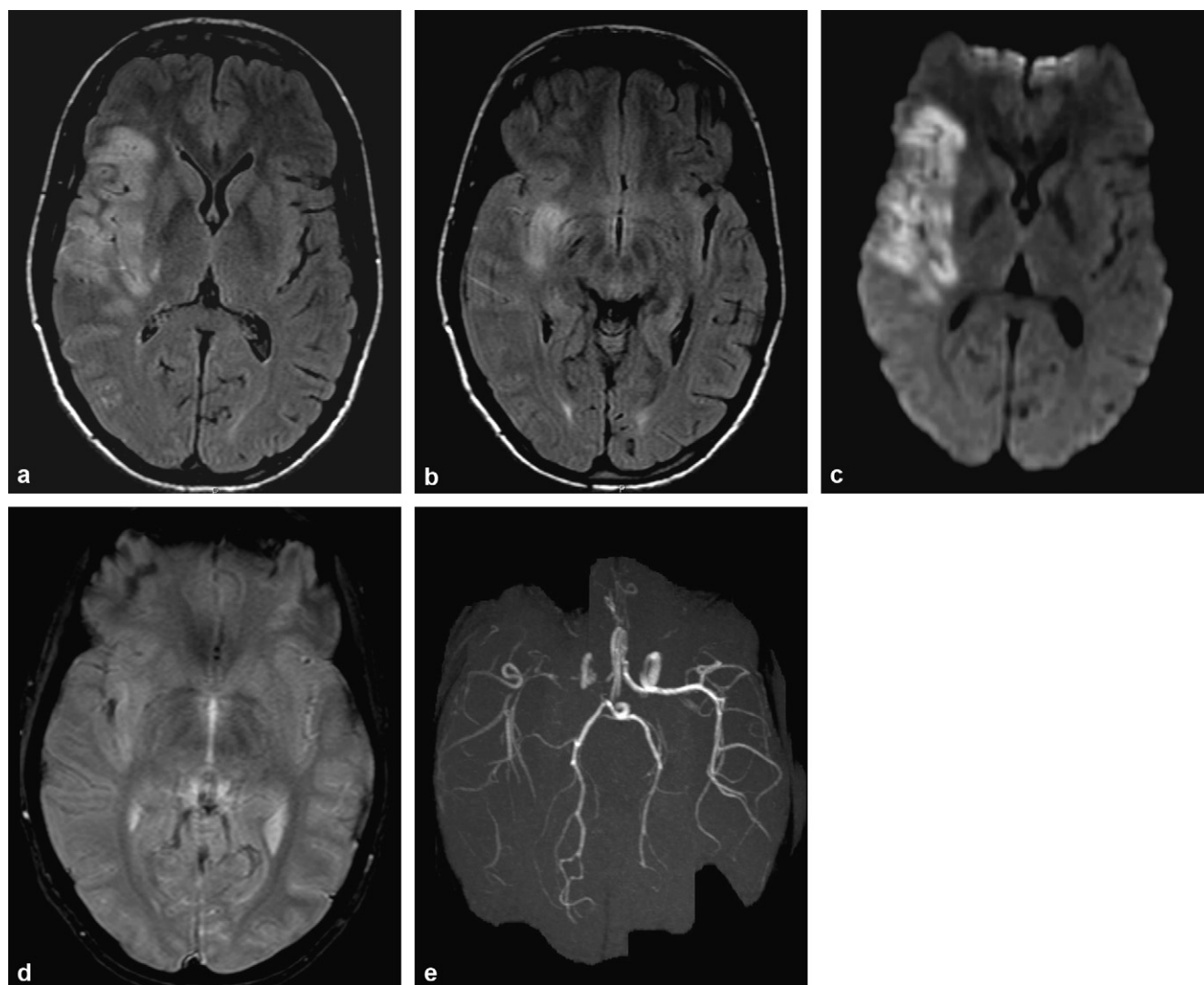


Figure 8. Ischaemic stroke on a vasospasm on day 8 of a neglected ruptured aneurysm in a female patient admitted because of confusion. a–b: FLAIR-weighted axial slices: right frontal-temporal hyperintensity; c: diffusion-weighted axial slice: hyperintensity of the right middle cerebral territory; d: T2*-weighted axial slice: low signal in the right lateral sulcus due to a thrombus or meningeal haemorrhage; e: MR angiogram: spasms of right middle cerebral artery.

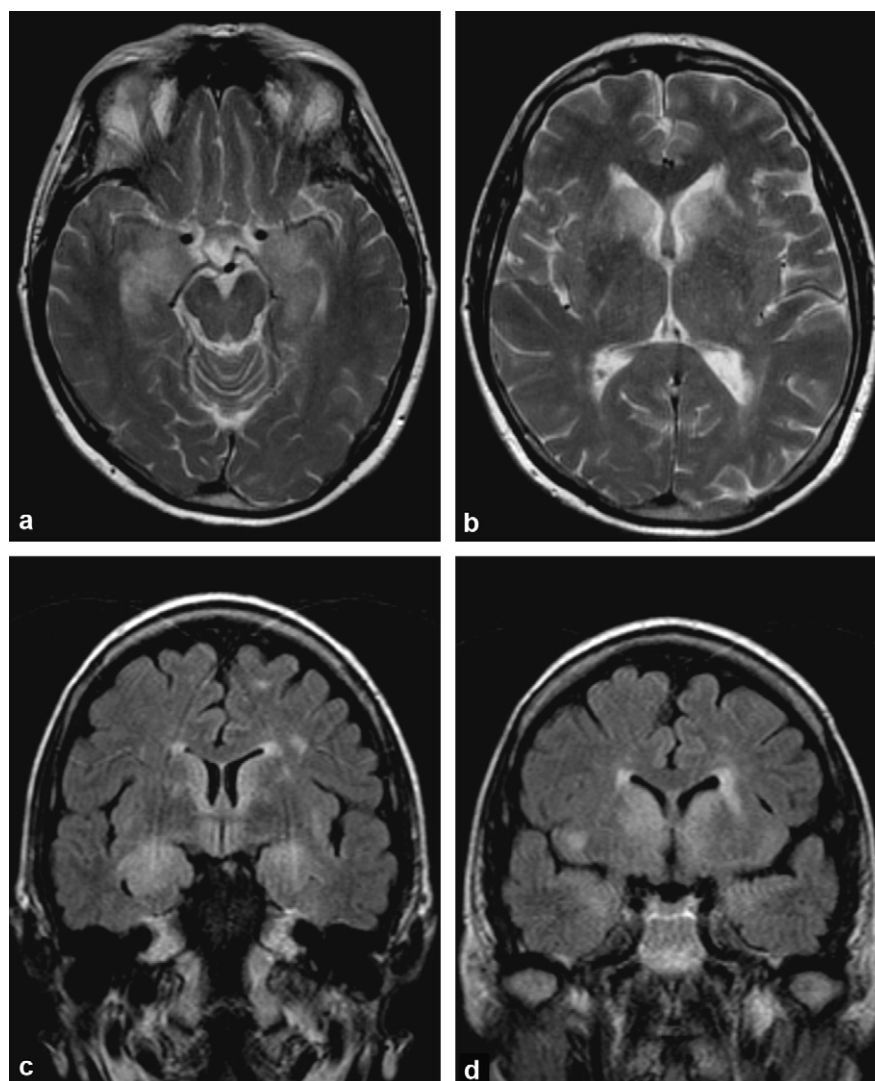


Figure 9. Paraneoplastic limbic encephalitis in a 71-year-old woman with cognitive impairment and acute aphasia. a–b: T2-weighted axial slices: bilateral medial temporal and caudate hyperintensity; c–d: FLAIR-weighted coronal slices: bilateral medial temporal cortical-subcortical and caudate hyperintensity.

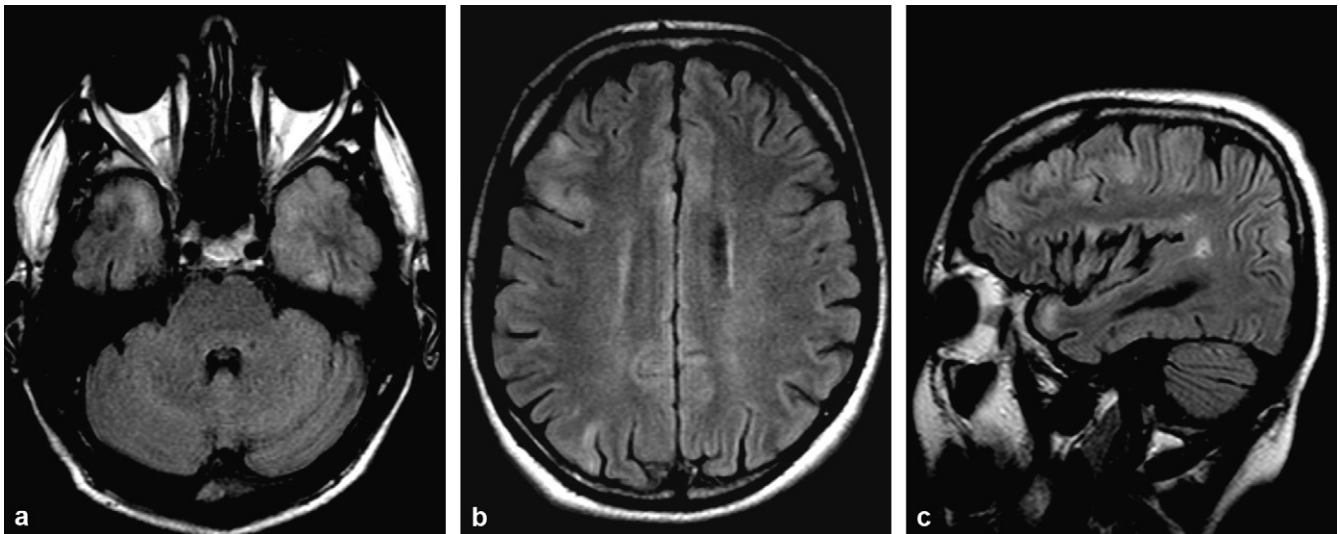


Figure 10. Syphilitic encephalitis in a 55-year-old man with subacute cognitive disorders. a–b: FLAIR-weighted axial slices: bilateral cortical hyperintensity predominantly at the temporal poles; c: FLAIR-weighted sagittal slice: anterior temporal and also frontal-parietal-occipital hyperintensity.

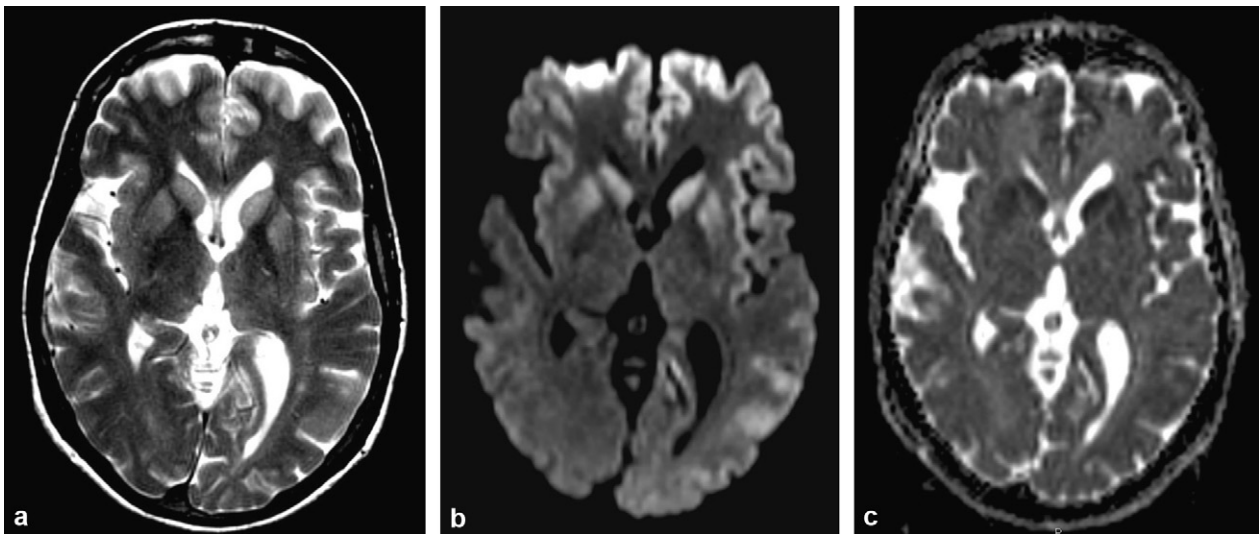


Figure 11. Creutzfeldt-Jakob disease, typical MRI appearance. a: T2-weighted axial slice: bilateral striatal hyperintensity; d: diffusion-weighted axial slice: bilateral striatal and cortical (bilateral frontal, left insular and posterior temporal) hyperintensity; c: axial apparent diffusion coefficient (ADC) mapping: ADC reduced.

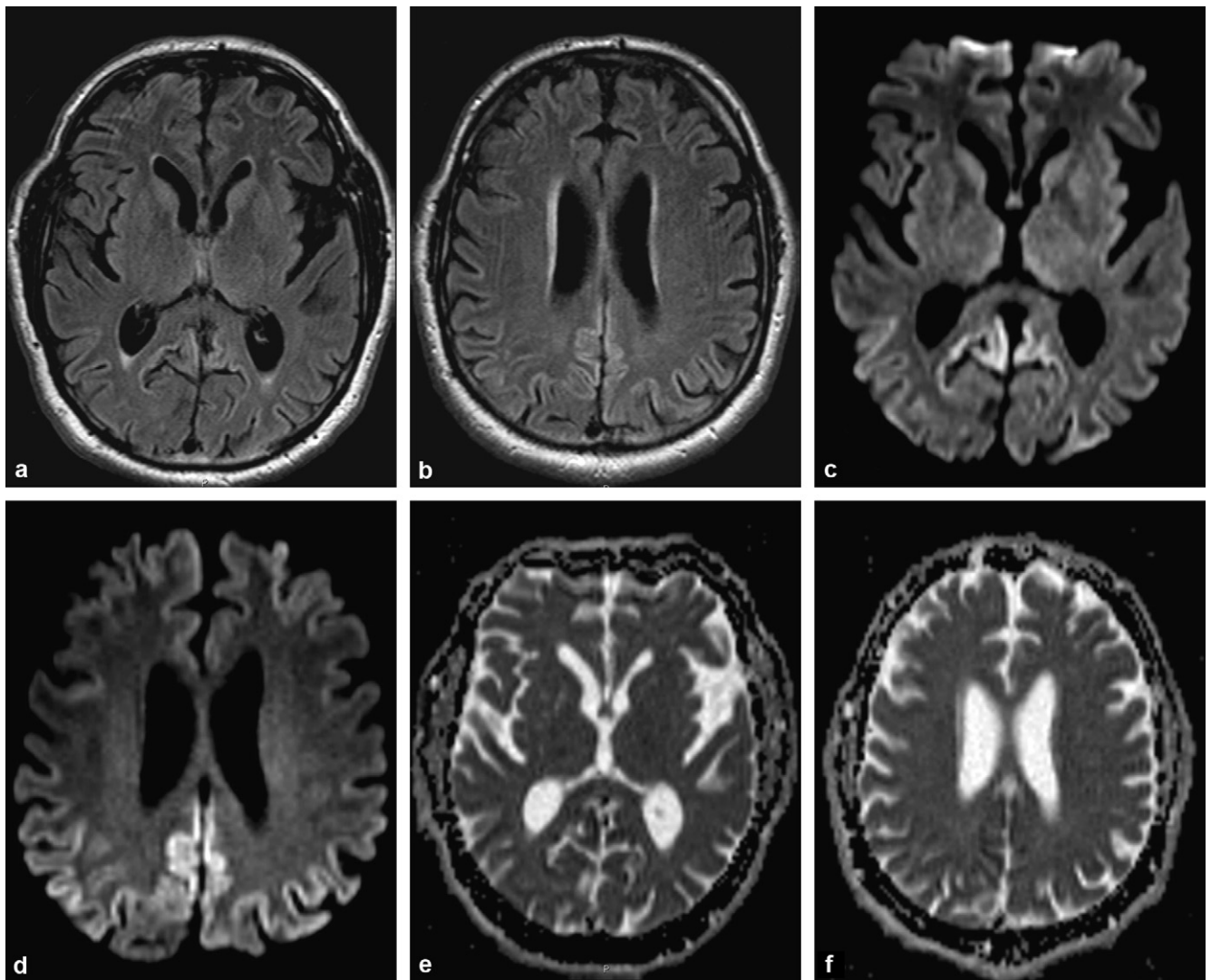


Figure 12. Creutzfeldt-Jakob disease in the early stages and exclusively cortical involvement. A seventy-three years old man with cognitive disorders which began several months previously. a–b: FLAIR-weighted axial slices: absence of distinct striatal or cortical hyperintensity; c–d: diffusion-weighted axial slices: bilateral parietal cortical hyperintensity with no striatal involvement; e–f: axial apparent diffusion coefficient (ADC) mapping: ADC reduced.

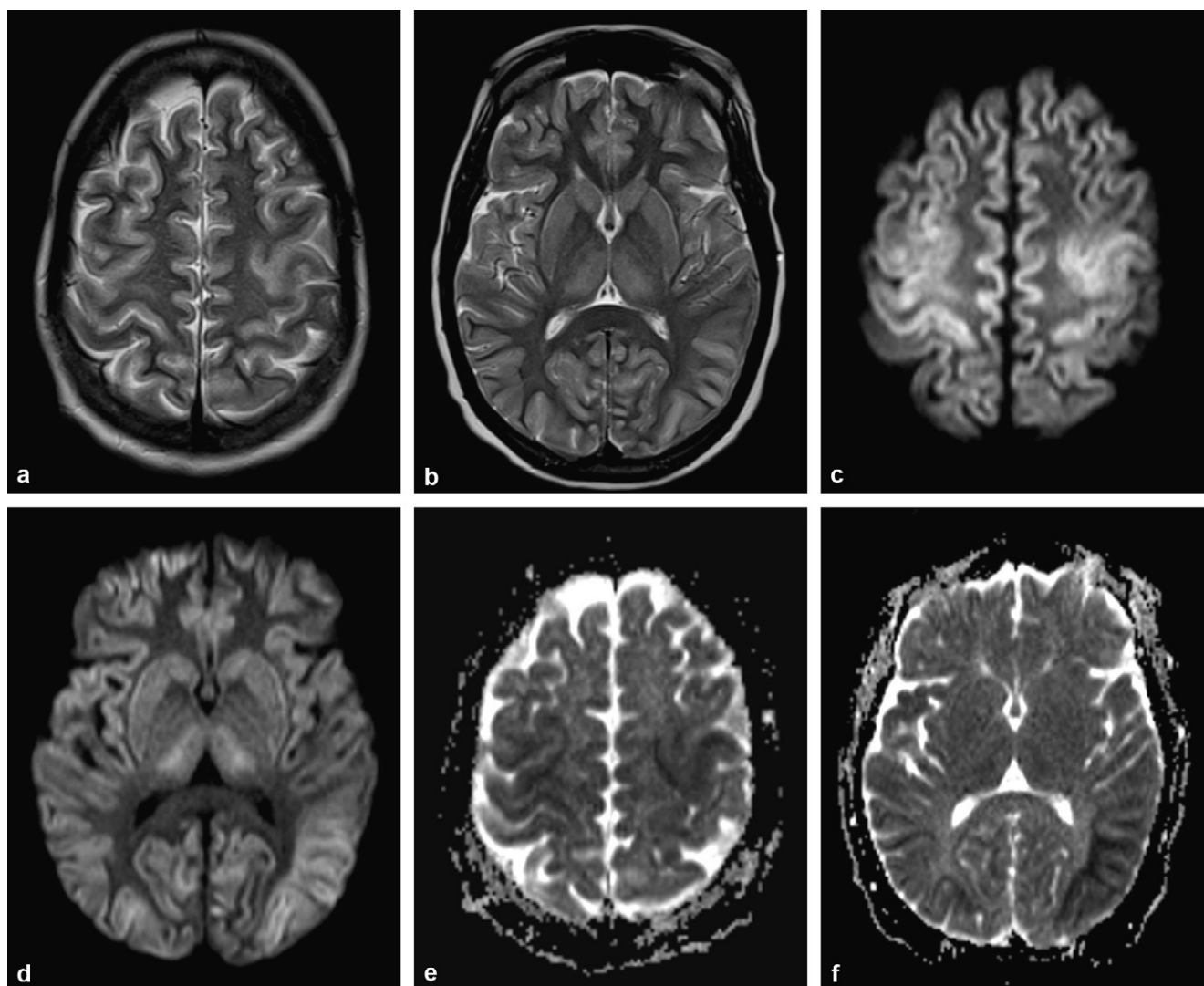


Figure 13. Hepatic encephalopathy in a 33-year-old alcoholic woman hospitalised for subacute cognitive disorders and coma. a–b: T2-weighted axial slices: bilateral diffuse cortical hyperintensity with basal ganglia involvement; c–d: diffusion-weighted axial slices: diffuse bilateral cortical, striatal and thalamic hyperintensity; e–f: axial apparent diffusion coefficient (ADC) mapping: ADC reduced.

the lesions resulting from status epilepticus which should be investigated by performing an EEG [18] (Fig. 15).

Exploration protocol when Creutzfeldt-Jakob disease is suspected

Exploration protocol when CJD is suspected:

- T2/FLAIR-weighting: grey matter hyperintensity (cortex and/or striatum and/or pulvinar). These sequences may be found wanting at the beginning;
- T1-weighting before and after gadolinium injection: enhancement eliminates a diagnosis of CJD;
- diffusion: the most sensitive sequence at the onset of symptoms. It will show a bilateral fall in the cortical and/or striatal and/or pulvinar ADC;
- T2*-weighting: there are never haemorrhagic changes; the presence of such changes points to a vascular condition, particularly deep vein thrombosis, which should be confirmed by MR venography.

Posterior reversible encephalopathy syndrome

Predominantly posterior (temporal-parietal-occipital) bilateral lesions suggest posterior reversible encephalopathy syndrome (PRES), the cause of which — hypertension, toxicity, chemotherapy, post-partum, etc. — must be sought. Clinical signs are a combination of headache, visual disturbances and epilepsy. Regression of the lesions in a few weeks will confirm the diagnosis.

MRI signs

The lesions related to vasogenic oedema are clearly visible as hyperintensity on a FLAIR sequence and primarily affect temporal-parietal-occipital subcortical white matter (Fig. 16). The deep white matter, the cortex, brain stem, cerebellum and spinal cord can also be affected [19–21]. T2-weighted spin echo sequences appear to be less

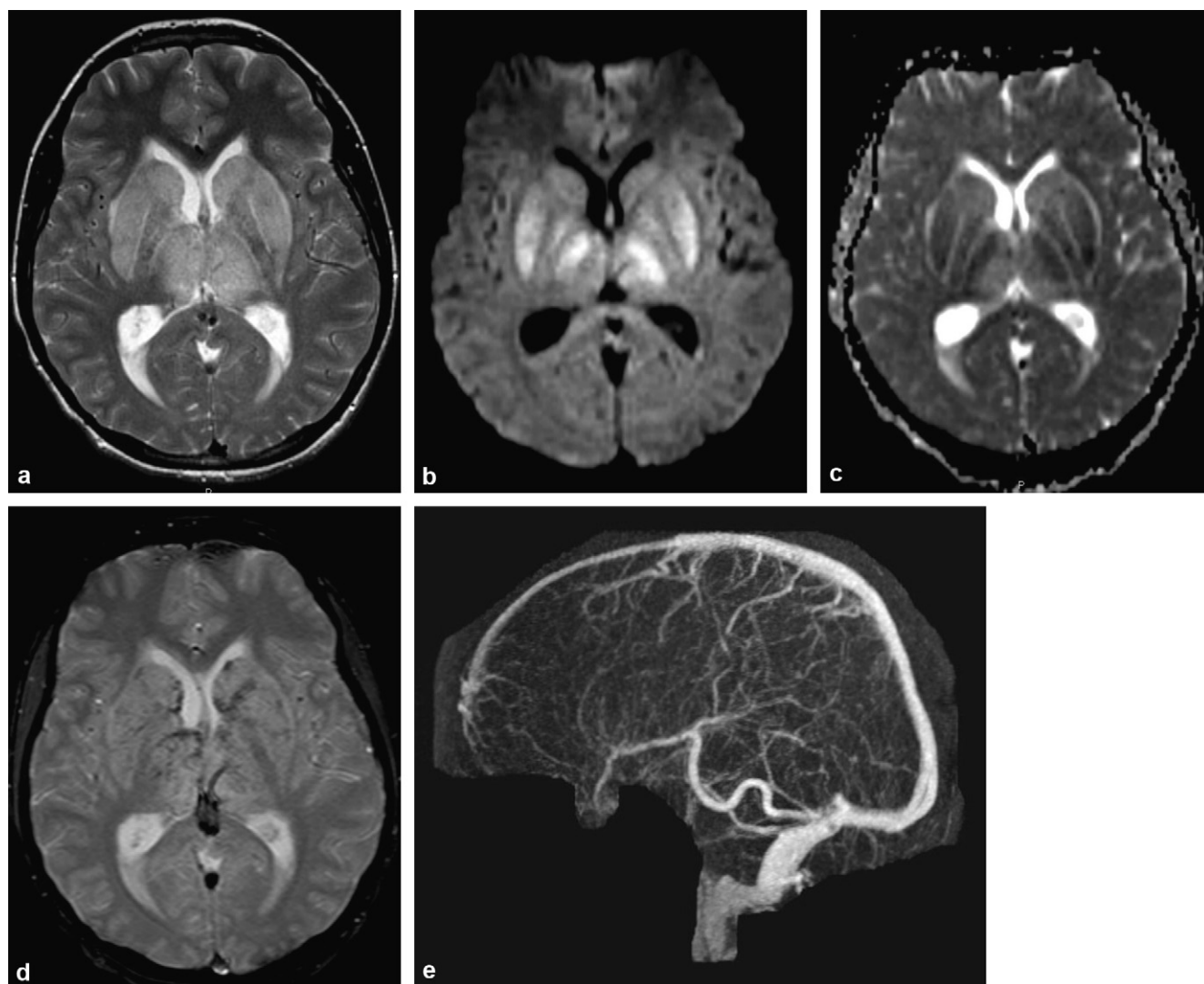


Figure 14. Deep vein thrombosis with venous infarction of the basal ganglia in a 25-year-old woman with subacute cognitive disorders and headaches. a: T2-weighted axial slice: bilateral hyperintensity of the basal ganglia; b: diffusion-weighted axial slice: bilateral hyperintensity of the basal ganglia; c: axial apparent diffusion coefficient (ADC) mapping: variable diffusion restriction within the basal ganglia; d: T2*-weighted axial slice: low basal ganglia intensity due to haemorrhagic changes; e: MR venography: the right thrombosed sinus is not visible.

sensitive. With T1-weighting these lesions present as discrete hypointensity and there is usually no enhancement. Haemorrhagic changes are possible and more frequently visible with susceptibility-weighted imaging (SWI) [22]. With subacute headaches, MR venography will eliminate a venous thrombosis (Fig. 16e). Cytotoxic lesions are sometimes present; they are accompanied by a fall in the ADC and enhancement is then possible. They have a poorer prognosis. When there is cytotoxic oedema, it remains limited and the lesions are less extensive than lesions associated with vasogenic oedema; if this is not the case the possibility of vasculitis can be considered and MR angiography should be performed to detect an arterial occlusion or stenosis (Fig. 17). Vasculitis can be distinguished from a reversible cerebral vasoconstriction syndrome (RCVS), sometimes associated with a PRES, by lesions that persist on a control MR angiogram at 3 months and by the different clinical context. Finally, when faced with predominantly posterior involvement of the white matter in a context of immunosuppression, progressive multifocal leukoencephalopathy (PML)

may be suspected, in which case definite hypointensity of the white matter with T1-weighting will be present (Fig. 18).

Exploration protocol where posterior reversible encephalopathy syndrome is suspected

Exploration protocol where PRES is suspected:

- FLAIR > T2-weighting: predominantly posterior cortical-subcortical hyperintensity;
- T1-weighting before and after gadolinium injection: absence, usually, of enhancement;
- diffusion: to look for associated cytotoxic lesions, which would indicate a poorer prognosis;
- SWI > T2*-weighting: microbleeds are often present;
- MR venography: to eliminate venous thrombosis in cases where headaches are occurring;
- MR angiography: to look for evidence supporting an associated RCVS.

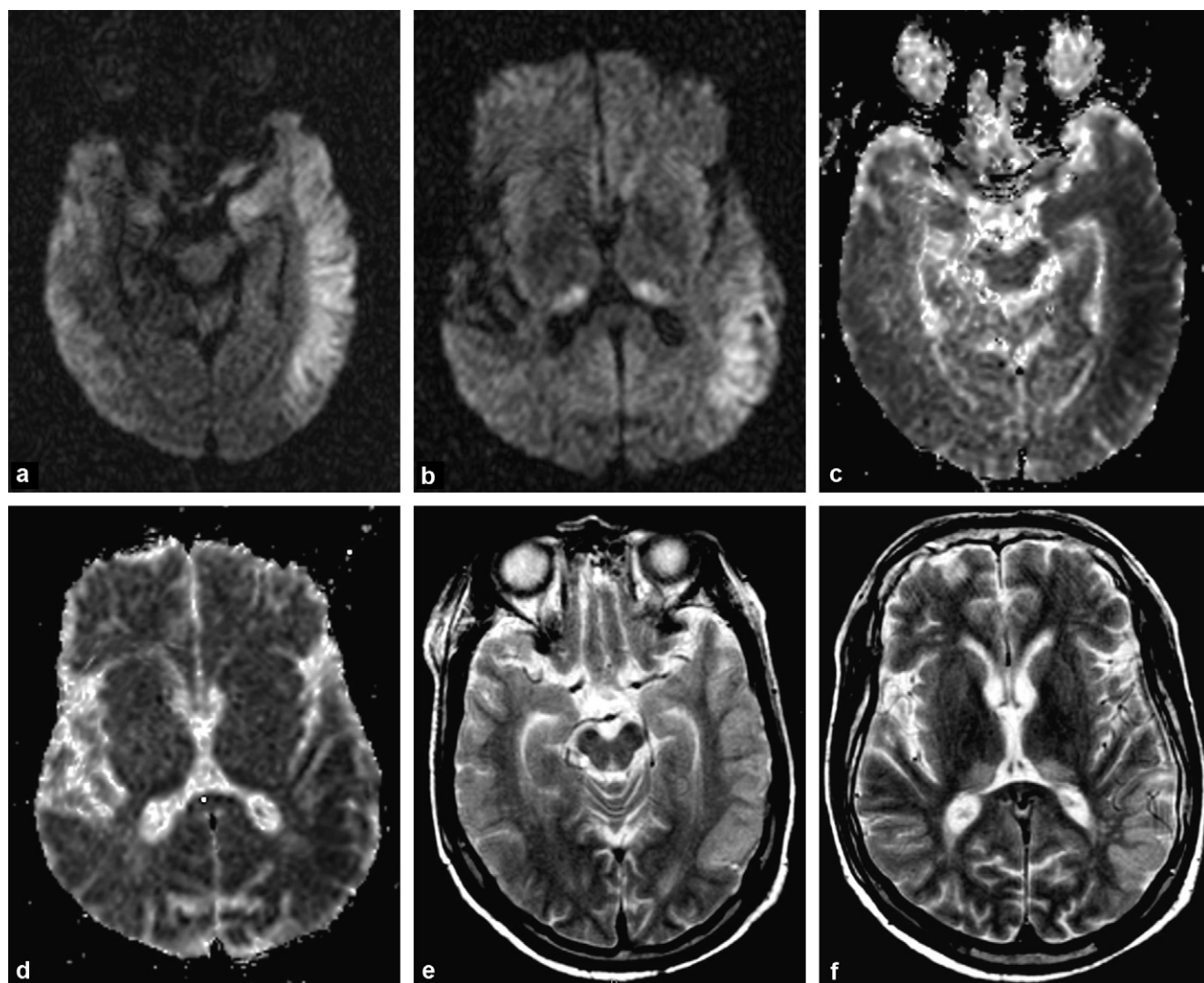


Figure 15. Status epilepticus lesions in a 32-year-old man in febrile status epilepticus: bilateral cortical temporal parietal and thalamic cytotoxic oedema. a–b: diffusion-weighted axial slices: bilateral temporal cortical and pulvinar hyperintensity predominantly on the left. c–d: axial apparent diffusion coefficient (ADC) mapping: ADC restriction; e–f: T2-weighted axial slices: bilateral cortical and pulvinar hyperintensity.

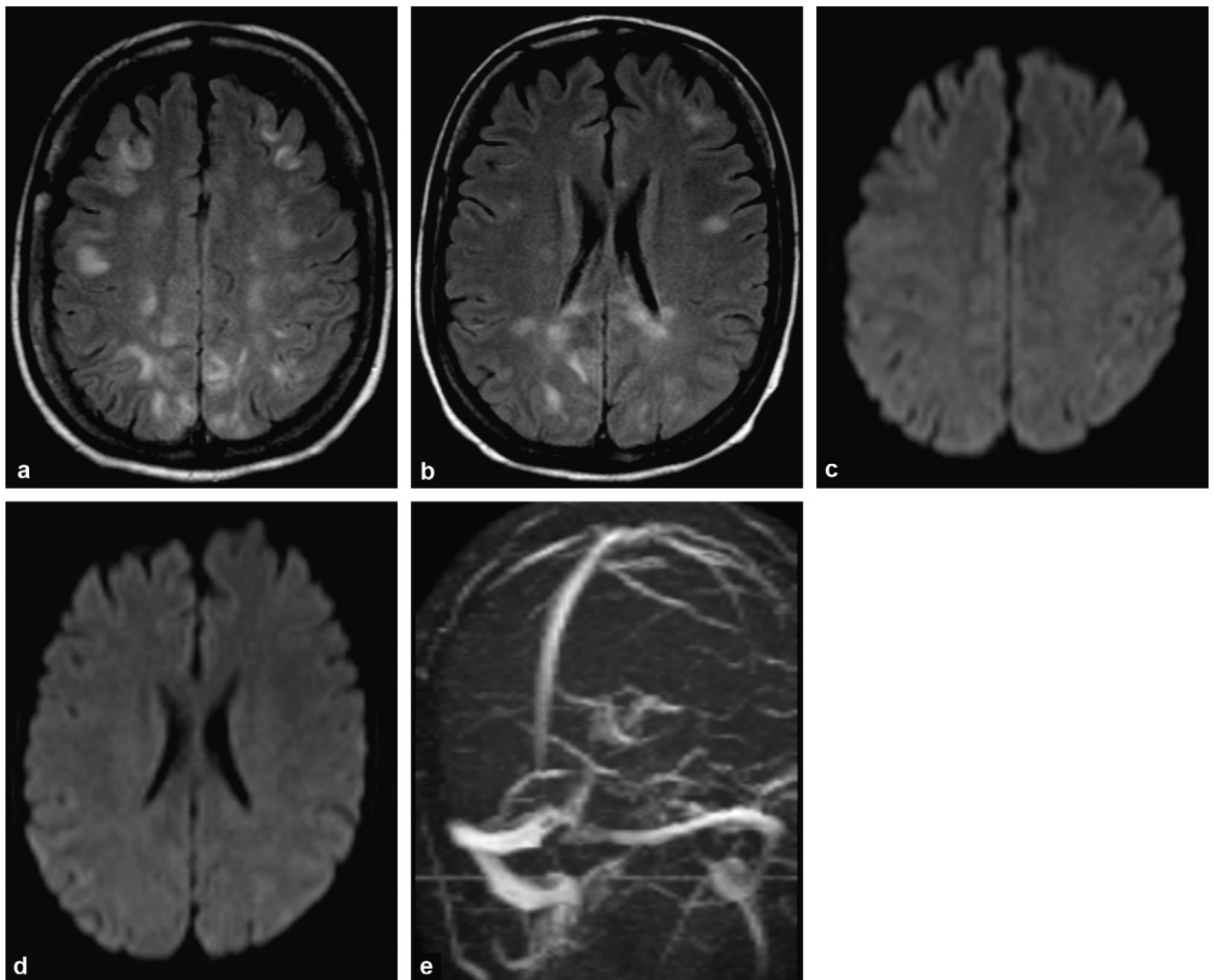


Figure 16. Typical PRES lesions in a 40-year-old female patient with epileptic seizures receiving tacrolimus for lung transplantation. a–b: FLAIR-weighted axial slices: bilateral diffuse cortical-subcortical hyperintensity, predominantly posterior and affecting the deep white matter; c–d: diffusion-weighted axial slices: no hyperintensity; e: MR venography: eliminates a venous thrombosis.

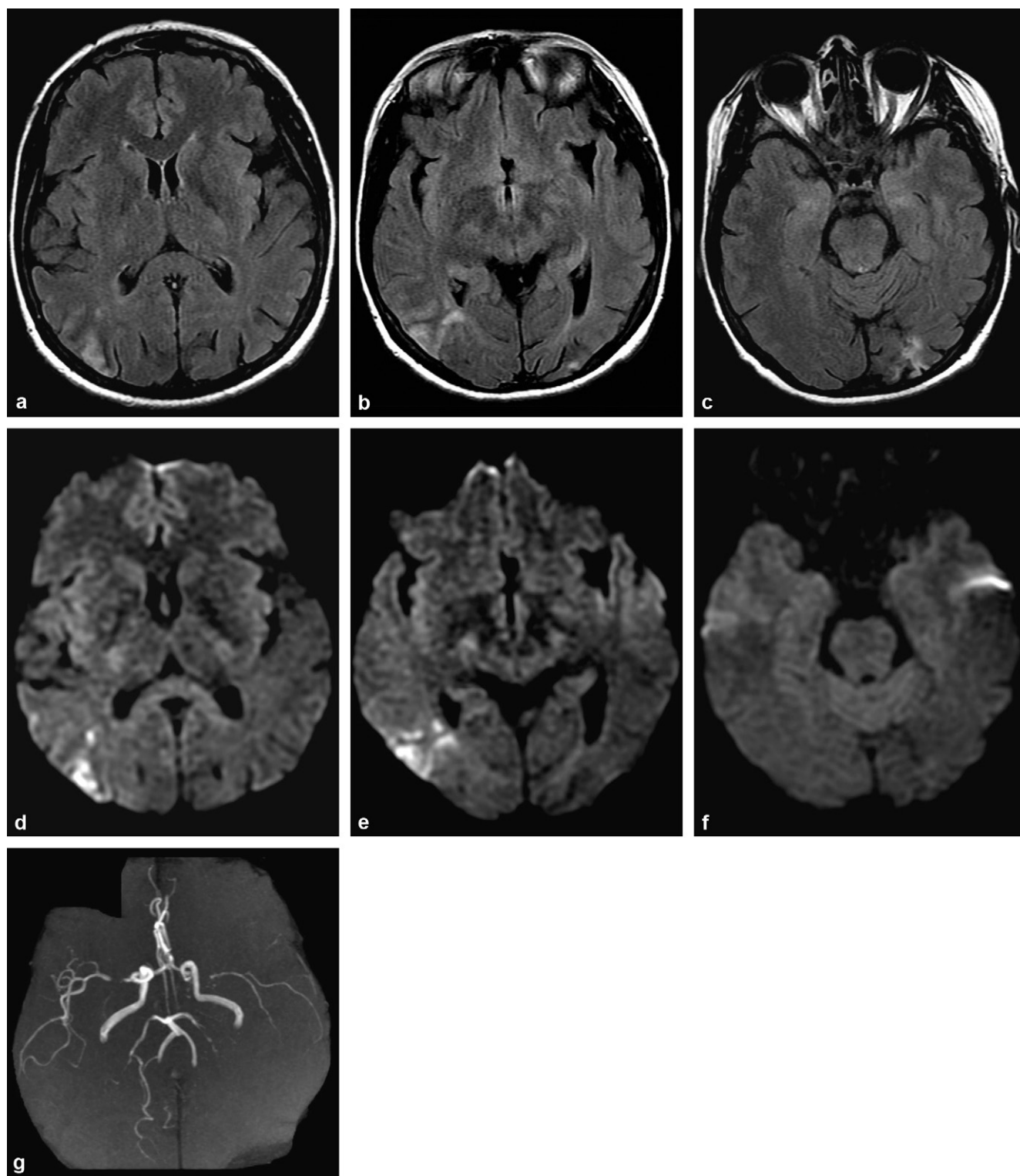


Figure 17. Vasculitis, with bilateral ischaemic lesions and multiple arterial stenoses in a 52-year-old woman with sicca syndrome. a–b: FLAIR-weighted axial slices: bilateral and posterior cortical-subcortical hyperintensity; d–f: diffusion-weighted axial slices: triangular hyperintensity in the right posterior cortical junctional zone suggesting recent ischaemia; g: MR angiography: bilateral multiple middle and posterior cerebral artery stenoses.

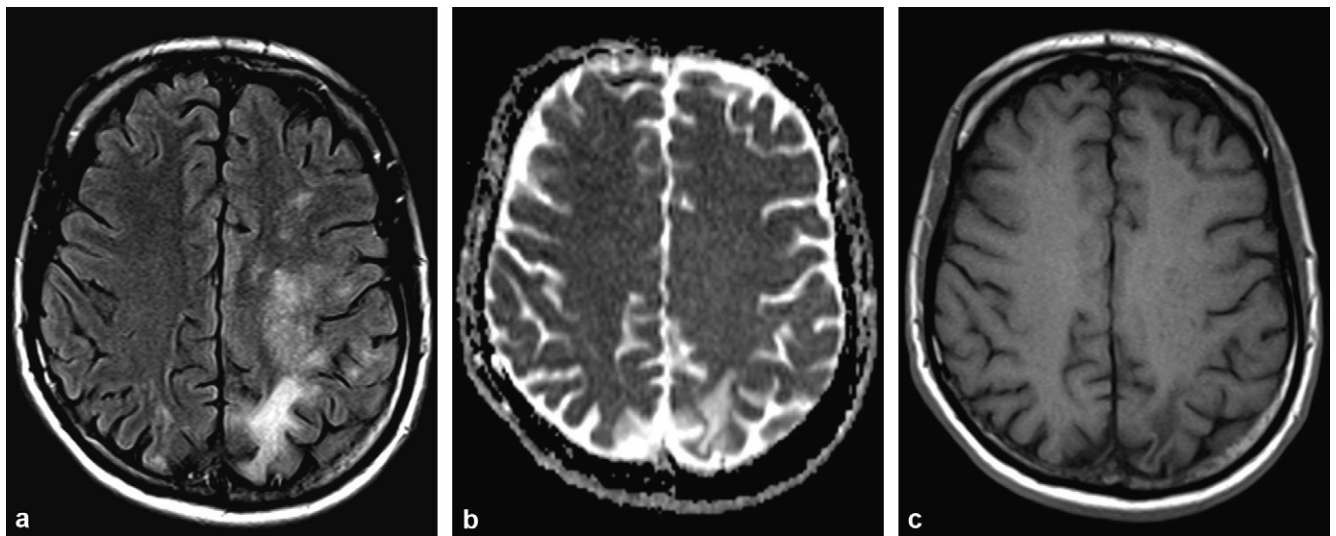


Figure 18. Progressive multifocal leukoencephalopathy in a 42-year-old man with ideomotor slowing and right motor deficit. Discovered following HIV positive serology. a: FLAIR-weighted axial slice: bilateral posterior cortical-subcortical hyperintensity predominantly on the left; b: apparent diffusion coefficient (ADC) mapping: no fall in ADC; c: T1-weighted axial slice: marked hypointensity of the left parietal white matter.

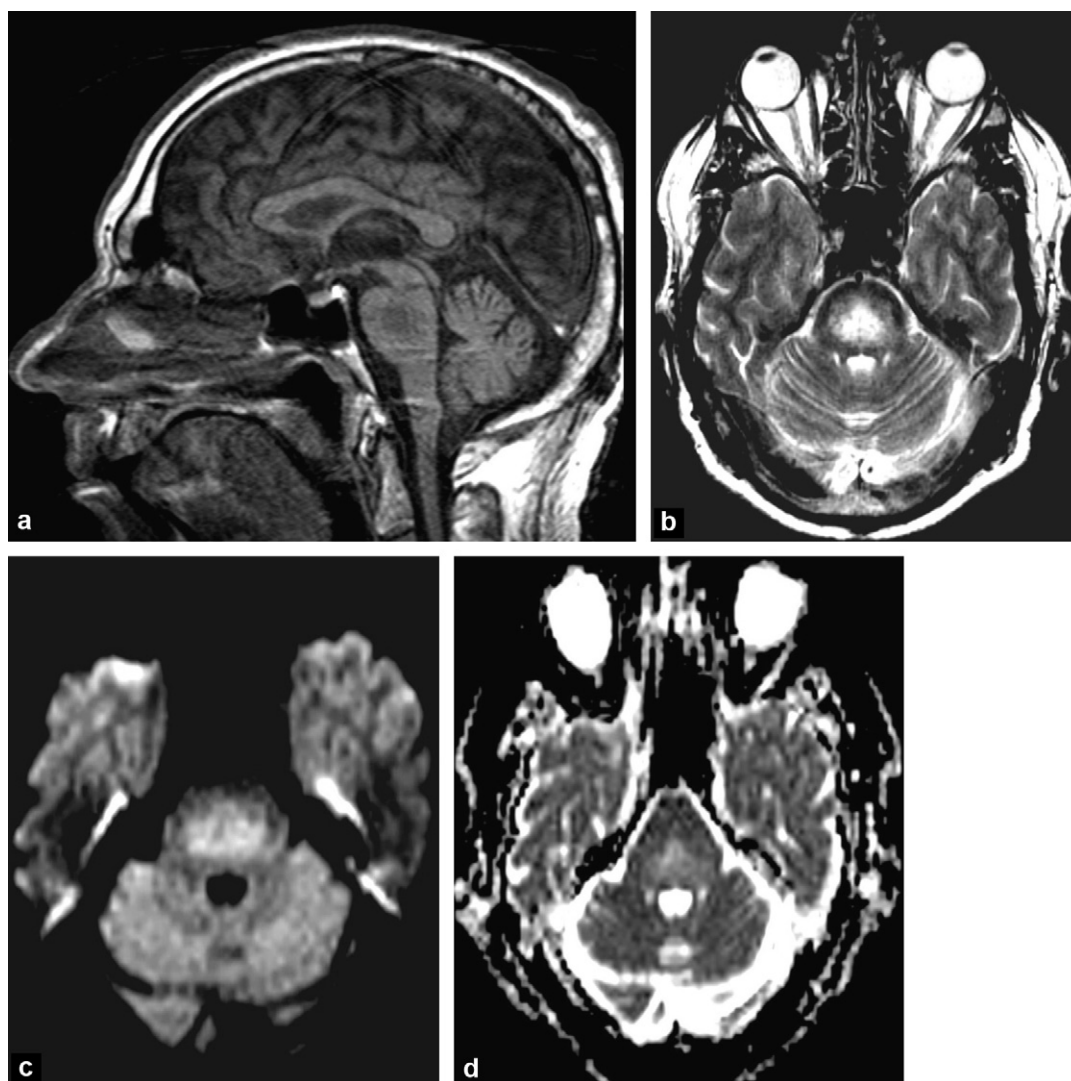


Figure 19. Central pontine myelinolysis in a 60-year-old alcoholic man presenting impaired balance and a cerebellar syndrome following hyponatraemia. a: T1-weighted sagittal slice: central pontine hypointense lesion; b: T2-weighted axial slice: hyperintense lesion sparing the peripheral fibres; c: diffusion-weighted axial slice: central pontine hyperintensity; d: axial apparent diffusion coefficient (ADC) mapping: no reduction in ADC.

Central pontine myelinolysis or osmotic demyelination syndrome

Central pontine oedema 2 to 3 days after rapidly restoring hyponatraemia in alcoholism and/or denutrition is very suggestive of central pontine myelinolysis (CPM). The clinical picture is very variable, typically of quadriplegia accompanied by pseudobulbar palsy and, in severe cases, coma.

MRI signs

Lesions of the pons appear hypointense with T1-weighting and hyperintense with T2/FLAIR-weighting. The corticospinal bundles and peripheral white matter are spared, which results in a trident-shaped appearance (Fig. 19). At an advanced stage, swelling of the pons is possible. Extra-pontine forms have been described with

involvement of the basal ganglia, thalamus, capsules and white matter [23,24] (Fig. 20). In the early days, there is ADC restriction on the diffusion-weighted images even before the oedema is visible in FLAIR/T2-weighting [25]. However, faced with coma and a lesion with restricted diffusion in the brain stem, brain stem ischaemia due to occlusion of the basilar artery should first be considered and a vascular exploration performed to confirm this hypothesis (Fig. 21). Some forms of PRES, linked to severe hypertension, can produce an isolated brain stem lesion [20]. In this case, the medulla and mid-brain are also affected. There is little or no diffusion restriction and MR angiography is normal (Fig. 22). Finally, non-expansive hyperintensity may be observed incidentally in the pons. It is then very likely to be due to white matter rarefaction lesions, the predisposing factors of which are mainly diabetes and renal impairment [26] (Fig. 23). Supratentorial leukoaraiosis is not always associated with this.

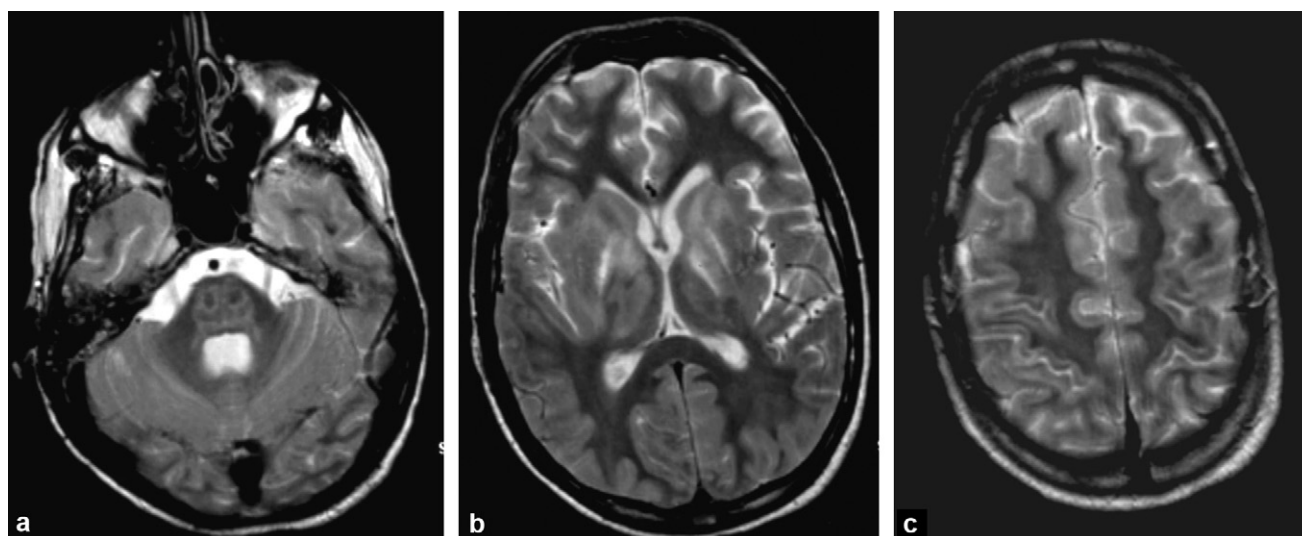


Figure 20. Central and extra-pontine osmotic myelinolysis in a 19-year-old young man with hyponatraemia in a context of trauma. a–c: T2-weighted axial slices: central pontine, basal ganglia, thalamic, internal and external capsule and subcortical white matter hyperintensity.

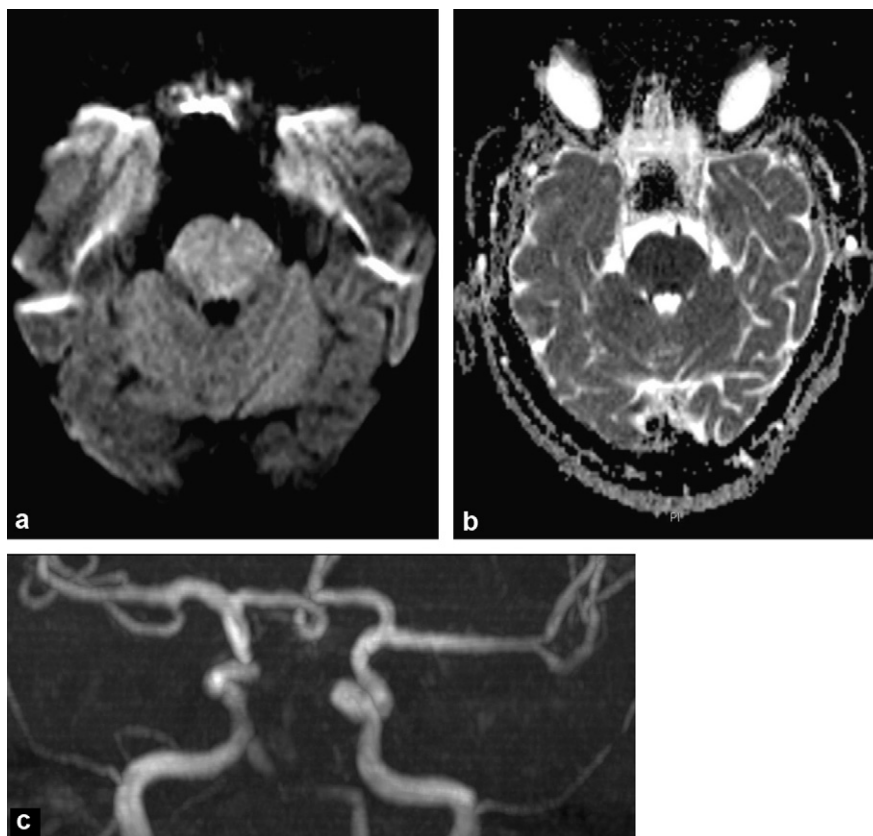


Figure 21. Brain stem ischaemia in a 50-year-old man hospitalised for headaches, malaise then coma. a: diffusion-weighted axial slice: hyperintense lesion of the pons with involvement of the peripheral fibres; b: axial apparent diffusion coefficient (ADC) mapping: fall in ADC; c: MR angiography: occlusion of the basilar artery.

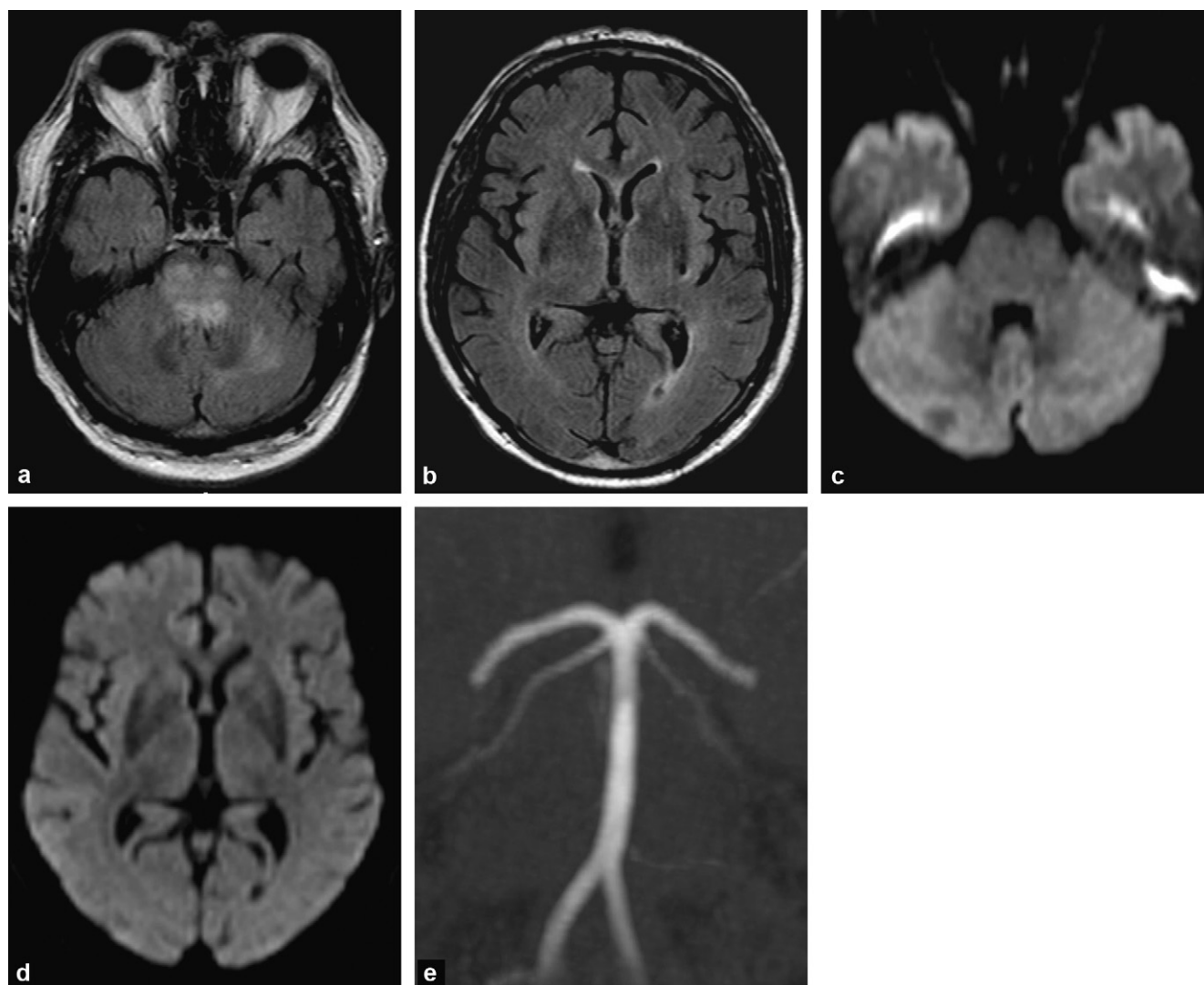


Figure 22. PRES in a 70-year-old man, who has had dysarthria and facial paralysis for 2 hours in a 24-hour context of acute hypertension. a–b: FLAIR-weighted axial slices: hyperintense lesions of the pons affecting the corticospinal bundles. No supratentorial lesions; c–d: diffusion-weighted axial slices: no abnormalities; e: MR angiography: basilar artery appears normal.

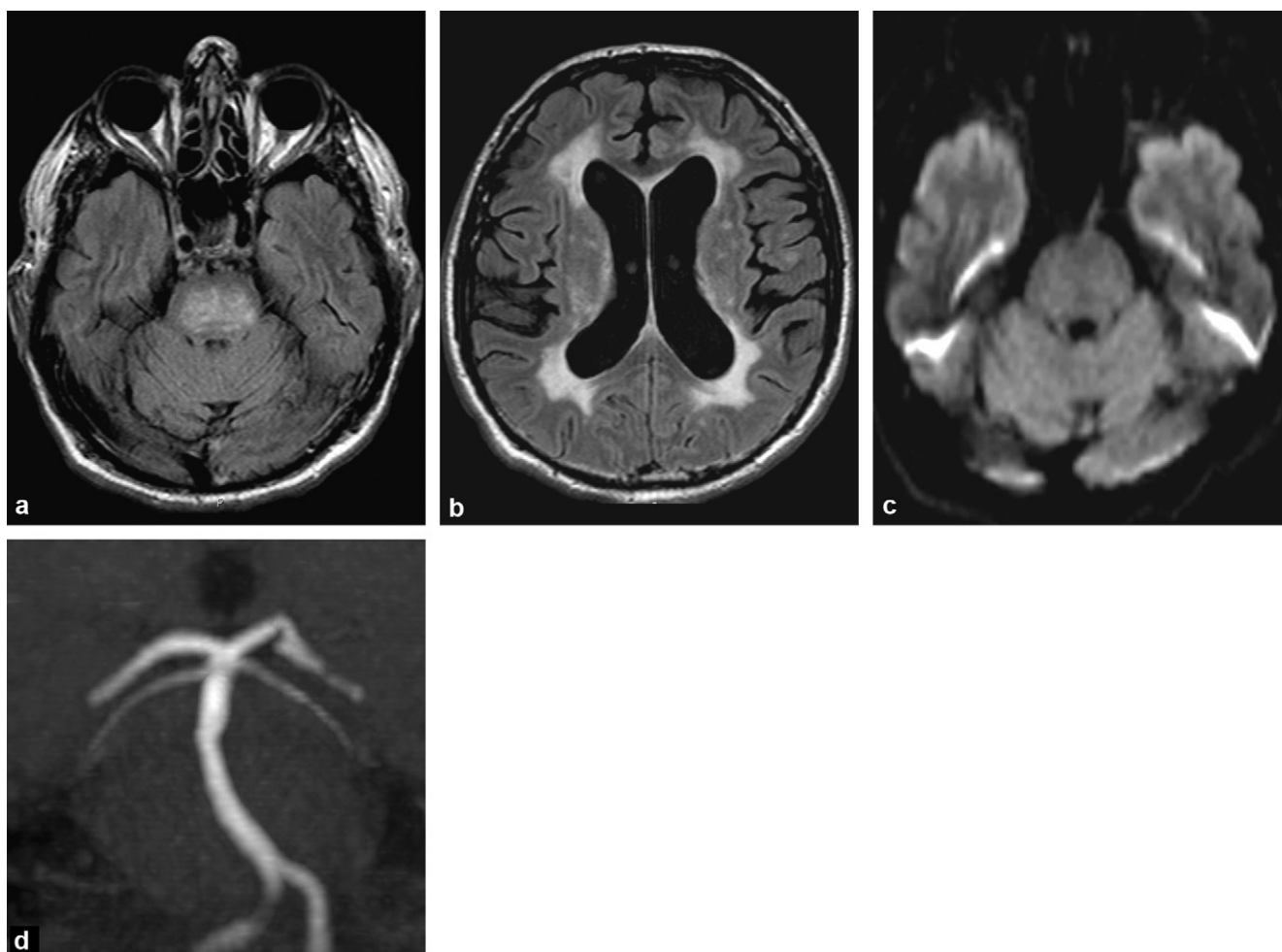


Figure 23. Pontine leukoaraiosis in a 71-year-old man with vascular risk factors. a: FLAIR-weighted axial slice: pontine hyperintensity; b: FLAIR-weighted axial slice: periventricular hyperintensity related to extensive leukoaraiosis; c: diffusion-weighted axial slice: no abnormalities; d: MR angiography: no occlusion of the basilar artery.

Exploration protocol where central pontine myelinolysis is suspected

Exploration protocol where CPM is suspected:

- T2/FLAIR-weighting: trident-shaped central pontine hyperintensity sparing the corticospinal bundles and peripheral fibres. Extra-pontine lesions are possible;
- T1-weighting before and after injection: central pontine hypointense lesion, contrast uptake may be seen;
- diffusion: the most sensitive sequence at the beginning. Diffusion restriction is present in the early stage;
- T2*-weighting: no bleeding;
- MR angiography: will eliminate occlusion of the basilar artery.

Conclusion

Through these five clinical conditions chosen from the vast field of central nervous infectious and metabolic disease, we have focused on the importance of:

- being aware of the clinical context;

- having good knowledge of the radiological signs;
- using specific sequences: diffusion, T2*-weighting, MR angiography, MR venography, perfusion and proton spectroscopy;
- to avoid the traps and make the best possible diagnosis.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

TAKE-HOME MESSAGES

General concepts

In metabolic and infectious diseases of the brain, MRI is the key examination due to the range of sequences available in addition to the conventional sequences: T2*, diffusion, proton spectroscopy, perfusion, MR angiography and MR venography.

Being aware of the clinical context (hypertension, neoplasia, chemotherapy, toxic immunosuppression, infection, status epilepticus, etc.) and laboratory data (hyponatraemia, etc.), as well as good knowledge of the signs of lesions, is essential for establishing the diagnosis.

Diagnostic pointers

Pyogenic abscess

This is revealed as a fluid-filled lesion with diffusion restriction, the walls of which are thinly enhanced. In proton spectroscopy, there is typically an amino-acid multiplet at 0.9 ppm, inverting with long TE.

- Not every fluid-filled lesion with diffusion restriction is a pyogenic abscess.
- The existence of definite T2* hypointensity points towards a haemorrhagic lesion.
- Exceptionally a necrotic metastasis may show diffusion restriction.
- An increase in CBV suggests malignancy.
- Finally, a toxoplasma abscess does not show diffusion restriction.

Herpes encephalitis

In the first instance, in a context of febrile confusion, bilateral, asymmetrical cortical-subcortical frontotemporal lesions should suggest herpes encephalitis.

- Associated striatal lesions suggest autoimmune or paraneoplastic limbic encephalitis.
- Unilateral involvement in the case of a suspected tumour, status epilepticus or an ischaemic stroke can require a perfusion sequence, MR angiography or EEG to help with the diagnosis.
- Where there is a subacute picture, syphilitic encephalitis should be considered.

Creutzfeldt-Jakob disease

This appears as bilateral lesions to the striatum and/or cortex and/or pulvinar thalamus, with diffusion restriction, in a context of progressive rapid deterioration of higher functions.

- An acute clinical picture will suggest rather anoxic lesions or hepatic encephalopathy, depending on the context.
- Pulvinar lesions are possible in status epilepticus.
- Haemorrhagic lesions of the basal ganglia point towards deep vein thrombosis.

Posterior reversible encephalopathy

This is suggested by predominantly posterior (temporal-parietal-occipital), asymmetric cortical-subcortical lesions in a particular context (post-partum, hypertension, chemotherapy or toxicity).

- Where there are headaches, MR venography will eliminate venous thrombosis.
- MR angiography will detect RCVS or vasculitis particularly if there are ischaemic lesions associated with diffusion restriction.
- With T1-weighting, definite hypointensity of the posterior white matter will point towards PML.

Central pontine myelinolysis

This is suggested by central pontine T2/FLAIR hyperintensity in the context of rapid correction of hyponatraemia.

- If there is associated diffusion restriction, brain stem ischaemia will be eliminated by MR angiography.
- In the face of extensive lesions affecting the medulla oblongata, pons and midbrain, hypertension should be investigated and a diagnosis considered of PRES.
- In the absence of symptoms suggesting a brain stem lesion, rarefaction of the white matter could be considered. Diabetes and renal failure are often the cause of this.

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